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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Treatment of Periodic Relapsing Catatonia

LEWIS DANZIGER and JOSEF A. KINDWALL
Wauwatosa, Wisconsin

In 1948, we described[2] a simplified method for the diagnosis and treatment of periodic catatonia. We showed then how this syndrome, so recalcitrant to treatment by such conventional methods as shock therapy and lobotomy, can be relieved by vitamins and thyroid extract given in adequate quantities over a sufficiently prolonged period. We showed also that the treatment may be started without the long preliminary studies first advised[9] and without giving dangerously large doses of thyroxin or thyroid extract. Our first report included experience with only one patient. We now describe our results with 30 additional patients, and a total of 44 attempts to control the disorder.

Theory

Since our first effort in this field, there has been developed a mathematical theory of periodic relapsing catatonia,[1] which describes the disorder and serves as a guide to the treatment. The theory may be described in non-mathematical terms as follows:

In the normal state, the thyroid and anterior pituitary glands, acting by the secretion of thyroid hormone and thyrotropin, are in nice balance. Thyrotropin stimulates the production of thyroid hormone, which inhibits the production of thyrotropin. Each hormone is lost or destroyed at a rate assumed to be proportional to the concentration of the hormone. If the thyroid is removed, thyrotropin proceeds, unchecked, at a rate which is assumed to be constant. When both glands are functioning normally and when, also, the losses are proceeding normally, the system is very stable. This stability is expressed in a basal metabolic rate (BMR) which is constant within the limits of experimental error. The BMR may not necessarily be at the optimum level for the patient's health, but it will show little variation.

Various abnormalities in the system may develop, for reasons now unknown. If the ability of either gland to respond to signals from the other becomes impaired and this abnormality persists, the system becomes unstable and the instability continues until it is corrected by appropriate treatment.

The theory predicts that the correct therapy is the administration of thyroid extract in doses sufficient to reduce the production of both endogenous thyroid hormone and thyrotropin to very low values. While this may permit some rise in the BMR, it will prevent the rate from falling below the critical and dangerous level. Stability is then maintained as long as the patient continues to take thyroid extract in sufficient amounts.

According to this theory there is an advantage in beginning treatment as suggested by Gjessing[9] when the BMR is near the low point of its cyclic course. This advantage is more than outweighed, from the practical, clinical standpoint by the time and effort required to determine the low point.

The theory implies, also, that some fluctuations in the patient's condition may be

From the Milwaukee Sanitarium.

expected after therapy is started. The duration of these abnormalities will depend on the amount of thyroid extract given, the location of the low point of the BMR-time curve, and the magnitude of the losses, which cannot be measured conveniently at present.

Very recently we have begun a study of the effect of treatment on the capacity of the thyroid gland to concentrate iodine, using the isotope I^{131}, after thyroid therapy has been given for several weeks. Our preliminary results indicate that a daily dose of 5 grains of thyroid extract will suffice to reduce the iodine uptake to less than one per cent of the normal value. This suggests that, under these conditions, the production of endogenous thyroid hormone is driven to zero, as required by the theory.

These predictions of the theory have been confirmed by our experiences.

Statistical Description of the Patients and Treatment

The clinical and biochemical aspects of periodic relapsing catatonia have been well described by Gjessing[9] and others[10,11,13,14] and need not be restated. We present here some statistical data on our 31 patients and in a later section give some illustrative case histories.

Age and Sex: Among the 31 patients treated by us, 6 were male. The distribution of ages at the time of the first attack was:

15-19 years	9 patients
20-24 years	8 patients
25-29 years	7 patients
30-34 years	2 patients
35-39 years	1 patient
40-44 years	2 patients
unknown	2 patients

Neither age at onset nor sex appeared to influence the results of treatment.

Duration of illness or relapse before the beginning of treatment, however, proved to be very important in determining the prognosis. The durations were distributed in the therapeutic attempts as follows:

less than 1 year	22 patients
1 to 2 years	3 patients
2.1 to 5 years	9 patients
5.1 to 10 years	3 patients
10.1 years or more	5 patients
unknown (many years)	2 patients

While the mathematical theory does not suggest that duration of illness will be an obstacle in the way of achieving stability in the thyroid-pituitary system, clinical judgment tells us that stability of the BMR will not suffice for recovery if the illness has persisted so long that permanent damage to the nervous system may reasonably be expected. The possibility must also be considered of strongly entrenched, not easily reversible reaction-patterns or habits in an illness of long duration.

Duration of period of the fluctuations in behavior could be determined in only 17 of the 31 individuals treated. For this determination, we had to rely on the history or on our own observations after the patient was admitted to the sanitarium. Gjessing has shown that the duration of each cycle can be determined best if the patient is first freed of all foci of infection and is then kept on a constant diet for a long time during which he receives no treatment. We were not able to meet these requirements, and were, in fact, more interested in determining that there was some period than in specifying its length. Where the period could be determined with reasonable accuracy, the distribution was as follows:

10 to 15 days	3 patients
20 to 21 days	2 patients
25 to 40 days	9 patients
40 to 84 days	3 patients

The mathematical theory indicates that, other factors being equal, the duration of the period will have a relation to the time required to achieve stability. Our group of patients is too small to throw any light on this question. All the patients with a period we could measure recovered except one, who had been ill for 6 years before treatment was started.

Precipitating Factors. No history of an important, possibly precipitating, event could be obtained in the cases of 15 of the 31 patients studied. Among the remaining 16, the factors discovered which might be considered precipitating were as follows:

Severe infection	4 patients
Severe injury	3 patients
Parturition	4 patients
Pregnancy	1 patient

Diet	1 patient
Death of fiance	1 patient
Stopped or reduced thyroid extract taken previously for other conditions	2 patients

Dose and Toxic Effects: Although, during the years covered by this report, we frequently reminded ourselves that the patients should be given the largest doses of thyroid extract or thyroxin which could be tolerated, we believe we have not attained this objective. While we said we would treat to the limit of tolerance, we forgot to ask ourselves the important question, "Whose tolerance?" In our thinking, we assumed it would be the patient's tolerance, but in practice we were often limited by the tolerance of ourselves, our colleagues, the referring physicians, or the relatives. Our results might have been somewhat better if we had had the courage to disregard expressed or implied criticism and alarm, and if we had fixed our attention firmly on the patients and on what doses they tolerate.

In determining the tolerance of the patient, certain special factors must be borne in mind. For example, tachycardia is not necessarily due to the thyroid extract administered. It may equally well be due to the psychosis, it may herald a relapse, it may be due to some temporary emotional disturbance. We have repeatedly seen the basal pulse rate fall from 120 per minute to 80 per minute in a few days or less, without any reduction in the dose of thyroid extract. We have often seen the pulse increase when the dose of thyroid extract was reduced.

Similarly, changes in weight must be regarded critically. We have seen many poorly nourished patients attain a normal weight as the appetite is stimulated by large doses of thyroid extract and vitamins and as the psychosis disappears. We have, of course, seen many obese patients lose weight. But it is rare for the weight to rise much beyond the normal or to fall much below the normal for the height, in those cases where the weight makes much change under thyroid therapy. In most cases, if the weight is abnormal in the beginning, it tends to become normal under therapy, whether this entails a gain or a loss in weight.

Edema is a symptom which we have learned to take fairly calmly. If the cardiac and renal functions are adequate, and if the plasma proteins are normal, edema which develops during thyroid therapy will disappear, in most cases within a few days, if the dose of thyroid extract is left unchanged.

Many patients develop moderately severe headaches as the BMR begins to rise. The significance of this symptom is unknown to us. The headache is controlled fairly well by aspirin and usually disappears in a few days.

Some patients complain of palpitation and mild dyspnea, especially upon exertion. In most cases, these symptoms pass off without treatment, if the dose of thyroid extract is left unchanged.

A high BMR during the course of thyroid therapy should not be taken too seriously, and is rarely an indication for reducing the dosage unless it is corroborated by clinical signs of intoxication. A high reading may be followed within a few days by one that is more acceptable, especially if there is some improvement, meanwhile, in the psychosis.

While we can now regard these symptoms calmly, they caused us much anxiety at first, and probably helped to limit the doses of thyroid extract given. The maximum daily doses of thyroid extract were distributed as follows:

2 grains	2 patients
4 grains	7 patients
5 grains	14 patients
6 grains	2 patients
8 grains	1 patient
10 grains	9 patients
12 grains	1 patient
15 grains	1 patient
16 grains	1 patient
20 grains	1 patient

A few patients were given thyroxin, orally or parenterally. The distribution of the maximum daily doses was:

2 milligrams	1 patient
3 milligrams	2 patients
4 milligrams	2 patients

The maximum daily dose was attained usually by several increments. The maintenance dose for those patients who recovered was usually near the maximum.

Vitamins: All patients receiving thyroxin or thyroid extract were given liberal doses

of the vitamin B complex orally, and sometimes also parenterally. In view of the increased requirement for vitamins produced by thyroid therapy,[16] we believe it is unwise to attempt to economize on these essential nutrients. The cost is negligible, even in a public hospital, in comparison with the economic losses occasioned by the illness.

Relapse: Relapses are known to have occurred after 11 successful therapeutic attempts. In 10 of these 11 cases, it is known that the relapse was preceded by a reduction in the daily intake of vitamins and/or thyroid extract or thyroxin, and in the remaining case no information is available. Of the 11 who relapsed, 9 returned for further treatment, and all who were given vitamins and thyroid extract or thyroxin recovered again.

Among the 9 treated twice were 4 patients who relapsed, recovered on further vitamin and thyroid therapy, and then relapsed again, apparently because of insufficient thyroid extract or thyroxin. Of these 4, three recovered for the third time and have remained well. The fourth was given thyroid extract for only a few days in her third admission because her family was persuaded by the patient that an immediate lobotomy was necessary to prevent suicide.

Two patients who are known to have relapsed did not return for further treatment. No information as to their condition has been obtained by us.

Insight: In our earlier publications on treatment, we have not laid stress on insight as a condition necessary to recovery. In thyroid cases we still believe that admitted insight is not necessary to recovery, but it is very desirable if the recovery is to be maintained. The patient who has recovered under vitamin and thyroid therapy will rarely, if ever, maintain his health if he does not admit the necessity of continuing the treatment at home, and if he does not maintain his intake of vitamins and thyroid extract or thyroxin at the level which has been found to be critical for him.

This places on the patient a burden which is not different from that borne by others with endocrine problems. As is true for patients with diabetes or adrenal insufficiency, failure of our patients to follow an appropriate therapeutic regime invites disaster.

Results of Treatment

As was the case with our other patients given thyroid therapy,[2] the percentage of patients recovering depends on the duration of treatment. The percentages have been calculated by the Dragstedt method of double integration,[5] (more properly called, perhaps, the method of double summation) used in our previous communication.[2] The essence of the method is this:

Patients exposed to the treatment (and therefore to the risk of recovery), for a certain number of days are counted. Of this group of patients, some will recover in the interval and some will not. One hundred times the number of recoveries divided by the number treated during this period of time is the percentage of success. For each other interval of time, the number treated and the number recovered will be unique, and will yield another percentage of success. The percentages may be calculated for any convenient number of different intervals of time, and the percentages may then be plotted against the corresponding times.

The results of these calculations, for the 44 therapeutic attempts, are shown in Table 1 and Figure 1. In these calculations, everything is ignored except the number of days of thyroid therapy and the percentage of success. Other important factors, such as the duration of the illness, the amount of thyroid extract given, and the use of acces-

TABLE 1

EFFECT OF DURATION OF TREATMENT ON THE PERCENTAGE OF SUCCESS

Number of days of thyroid therapy	Number of patients treated for this number of days	Number of patients recovered within this number of days	Percentage of success within this number of days
10	44	2	4.5
20	44	3	6.8
30	41	7	17.1
40	39	9	23.0
50	39	13	33.3
75	37	18	48.6
100	35	23	65.7
150	33	26	78.8
360	30	27	90.0

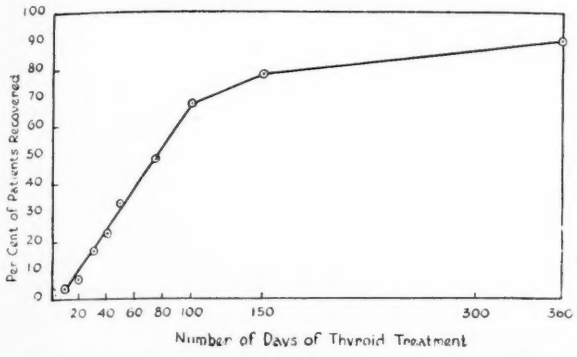

Figure 1. Per cent of patients recovered, calculated by the method of Dragstedt and Lang,[5] plotted against the number of days of thyroid therapy. The response is linear in the first 100 days of treatment, but 100 days suffices for only about two thirds of the recoveries.

sory types of treatment, such as shock therapy, are neglected in these calculations.

It is seen from the table and graph that the response to treatment in the first 100 days is a linear function of the time. The response-dose curve thereafter rises more slowly, and approaches a limiting value of about 90 per cent success. About two thirds of those patients who were treated for 100 days recovered; but 360 days of treatment were required by the patient who recovered last.

Five of the 27 recoveries occurred within 30 days or less, and three occurred with 11 days or less. These recoveries were so rapid and dramatic that it seems likely the treatment was started just when the patient was beginning a spontaneous improvement. In these cases, also, the dose of thyroid extract and the length of the period must have been such that there was time for the system to become stable before the next relapse was due. No patient in this fortunate group received more than 5 grains of thyroid extract daily as his maximum dose; however, this means only that they recovered before there was time to indicate the necessity for larger doses, and does not imply that small doses are more effective than large ones.

When a patient recovers so promptly, there usually is no reason to consider other forms of treatment. For the less fortunate, it is necessary to make important decisions and sometimes concessions to insure sufficiently long treatment. Should shock therapy be used for the purpose of giving temporary relief, or to make the patient more tractable? Must shock therapy be used to satisfy the relatives, or the referring physician or, in rare instances, the patient? For how long is it justifiable or expedient to resist the demands of various persons that the patient be "given some specific treatment," or "some treatment," or "something besides pills, which he could take at home just as well"? These are difficult questions for which we have no ready answers except in statistical terms, which can never satisfy those interested in single individuals.

Superficially, our results would seem to favor confining treatment to thyroid extract and vitamins. Fifteen therapeutic attempts were completed without the use of any other therapy; of these attempts, 13 were successful. The two which were not successful were stopped before the 100th day. To score such a percentage of success among patients treated for at least 100 days leaves nothing to be desired, except speedier results.

In the other 29 attempts at treatment, thyroid extract was given with some combination of shock therapy, lipotropic factors, carbon dioxide, and adrenal cortex extract. The various combinations were tried to give patients temporary relief, to attempt to increase the rate of improvement, or for other reasons. Of the 29 attempts, 7 were discontinued before the 100th day of thyroid; of the remaining 22 attempts, 15 were successful. Five of the 7 failures, in the group treated for at least 100 days, occurred in patients who had been ill for 5 years or longer; the other two failures occurred in a single patient. One of the failures, who had been ill for at least 15 years, was apparently well, although lacking in admitted insight, when the responsible relative insisted that treatment be stopped; this patient promptly relapsed and was counted as a failure.

From the comparison of the two groups (one given only thyroid extract and vitamins, the other given thyroid extract, vitamins and conventional therapy), it seems to us that conventional therapy does no harm and may do some good. Conventional therapy was used by us, at least in some cases, because we thought it would be helpful, if not to the patient, then at least to the nurses. The patients receiving combined therapy impressed us as more difficult than those re-

ceiving only thyroid extract and vitamins; those in the combined therapy group were more disturbed, or had been ill longer, or showed no early improvement on thyroid extract. Two of them had periods of a year or longer in duration. Some were given shock therapy before the diagnosis of periodic relapsing catatonia was made, and the treatment was continued for a short time after thyroid therapy was started.

In some cases, shock therapy acted like a strong, rapid but short-acting sedative. Some of these patients appeared to improve rapidly as the first few shock treatments were given, only to relapse as the treatment was continued. This important phenomenon of relapse during treatment is nearly diagnostic of periodic relapsing catatonia.

Some other patients relapsed immediately after a short course of treatment, which had apparently produced striking improvement; in such patients, the diagnosis is not quite as certain but it should be considered seriously. Relapse during or immediately after treatment is not, therefore, a discouraging sign but a strong indication that success may be expected from adequate thyroid therapy.

Those who recovered on combined therapy seemed just as well as those who recovered on thyroid extract and vitamins alone. This was true, also, of those who recovered on thyroid extract and vitamins, alone or in combination with other therapy, after having had very prolonged and intensive shock therapy here or elsewhere before the thyroid was tried. Steinfeld[19] has described a very striking example of this observation. His patient (F.Q.) had some 200 ECTs before she recovered from her periodic catatonia under combined thyroid and shock therapy. She then was able to return to college, where she did good work; since then she has been gainfully employed as a secretary.

Various investigators[4,6,7,8,12,15,17,20] have expressed interest in liver function in mental disorders and their publications led us to try lipotropic factors in 7 patients. In this group, two stopped thyroid therapy before the 100th day. Of the remaining five,four recovered. The patient treated unsuccessfully had been ill for five years. Some patients in this group had shock therapy also.

Four attempts were made with adrenal cortex extract combined with vitamins and thyroid extract. Of the three attempts which were continued to the 100th day of thyroid therapy, one was successful. The two failures occurred in a single patient.

The duration of illness or relapse affected the prognosis as follows, in patients who had at least 100 days of thyroid therapy:

When the duration was 2 years or less, 20 patients recovered of the 21 treated; when the duration was 2.1 to 5 years, 5 patients recovered of 6 treated; when the duration was more than 5 but less than 10 years, one patient recovered of 3 treated. Two other patients, ill for 10 years or longer, were treated for 100 days or more without recovery; 2 other patients, ill for "many years" were treated for 100 days without recovery.

While the failures weigh heavily on us and command much of our attention, we should emphasize that no patient appeared to be made any worse by thyroid therapy, and that nearly all the patients counted as failures were improved or much improved when discharged. Some of those counted as failures were able to return to work for a time.

Finally, it should be emphasized that no patient was taken off thyroid therapy because of our wishes, either before or after the expiration of the 100 day period. We discontinued treatment only because of circumstances beyond our control. We have been at some pains to give the difficult as well as the easy cases as much opportunity to recover as we were permitted to give them, and to include both the difficult and the easy in our statistics.

Illustrative Case Histories

No single patient may be expected to exhibit all the possible symptoms of periodic relapsing catatonia or to illustrate all the difficulties which may be encountered in treatment. The following histories are selected because they present some of the more important aspects of the problem.

The first patient demonstrates the dramatic onset often seen after an infection, a very short period with several relapses under conventional treatment, the absence of any laboratory signs of thyroid deficiency, and recovery under vitamins and thyroid ex-

tract after other types of treatment were abandoned.

This patient, K.M., was born in 1930. Her early life was uneventful. She had always been considered very stable and well adjusted, and was an excellent student. In her final year in college at age 22, she developed infectious mononucleosis, from which she appeared to recover without difficulty. Four months after the infection she complained of fatigue, which persisted for three weeks. At the end of this period, the patient suddenly became overtalkative, restless, delusional and, within 24 hours, combative.

On admission to the sanitarium, less than 24 hours after the onset of restlessness, the patient appeared to be in a catatonic excitement. Liver function tests (bromsulphalein retention, cephalin flocculation, prothrombin times and plasma proteins) were normal. Blood cholesterol was 247 milligrams per cent. The lowest BMR, obtained during the excited period, was plus 12. There was nothing in the physical examination to suggest a thyroid disorder.

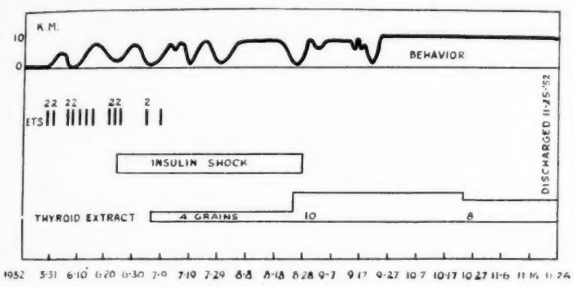

Figure 2. Response of the patient K.M. to various types of treatment. Upper curve shows behavior, measured in arbitrary units; a dip in the curve indicates a relapse. Second line shows ECTs; the figure 2 indicates a double treatment for that day. Daily dose of thyroid extract, in grains, is indicated.

The patient's various treatments and her stormy course are summarized in Figure 2. Relapses during period of shock therapy were very striking. Insulin and electroshock therapies, singly or together, had no apparent effect on the course of the illness, and were abandoned after the patient had had 19 electroshock treatments and 33 insulin stupors. The final relapse ended on the 81st day of vitamin and thyroid therapy, and 25 days after the daily dose of thyroid extract was increased from 4 grains to 10.

Later the maintenance dose was established at 6 grains of thyroid extract daily. The patient has been well since September, 1952.

Another patient is of interest because of the family history, and presence of liver damage and the reluctance of the referring internist to permit thyroid therapy.

This patient, M.B., female, was born in 1921, a member of a pair of twins, said to be identical. The twins were married at about the same age (22 years) and each gave birth first to a single child and later to a set of twins. Soon after the birth of her twins, our patient's twin became psychotic and committed suicide.

Our patient had what was called a depression in 1946; she recovered under combined electroshock and insulin shock therapy, given elsewhere. She was delivered of her twins on October 9, 1950. The next day she was irritable and soon became depressed. She improved rapidly under electroshock therapy in another sanitarium, but relapsed after a few days when she received the news of her sister's suicide. The patient improved again, and was allowed to go home for a visit at Christmas. There she attempted suicide by barbiturates. She was given more electroshock, with apparently good effect and was discharged. At home she relapsed quickly, and drank carbon tetrachloride. She was returned to the original sanitarium, improved again under more electroshock, and was discharged in a few days. After four weeks at home, she drank more carbon tetrachloride and was admitted to the Milwaukee Sanitarium.

It was then learned that the patient had been given some thyroid extract by her obstetrician during the last pregnancy. This medication had not been continued by the patient, and had not been administered during the psychosis. Physical examination on admission revealed nothing of interest. The blood cholesterol was 211 milligrams per cent. BMR and liver function tests were done later.

The day after admission, patient vomited bloody material. The catheterized urine showed albumen and many casts, and the temperature began to rise. The patient was transferred to a general hospital for treatment of presumed liver and kidney damage due to the carbon tetrachloride.

On the patient's return to the sanitarium, a week later, the bromsulphalein retention was 24 per cent, the prothrombin time was 50 per cent, but the cephalin flocculation was negative and the plasma proteins were 5.8 grams per cent. The non-protein nitrogen of the blood was 36 milligrams per cent. The BMR was plus 1. The liver and kidney functions improved rapidly under lipotropic therapy, but the patient remained depressed and suicidal, with delusions of reference, and later became stuporous.

The patient was given the following conventional treatment: lipotropic factors, orally, from April 21, 1951 to May 14, 1951; electroshock, 13 sine wave treatments, from May 29 to July 2; and 4 sine wave treatments from August 7 to August 16; insulin shock, 8 stupors, from May 29 to July 20; and 4 stupors, from July 31 to August 17.

The poor effect of this treatment, and of the electroshock given previously, may be seen from the following table of relapse dates.

October 10, 1950—Onset of illness.
November 30, 1950—News of sister's death.
December 25, 1950—Attempt with barbiturates.
March 7, 1951—Attempt with CCl_4.
April 14, 1951—Attempt with CCl_4.
June 11, 1951—Relapse during shock therapy.
July 29, 1951—Immediately after shock therapy.
August 22, 1951.

The referring internist agreed after the sixth relapse that conventional therapy would not suffice. Thyroid extract, in a daily dose of 5 grains, was started on August 2, 1951. The seventh and final relapse ended on September 1, 1951. The patient has continued since then on 5 grains of thyroid extract daily, with vitamins, and has remained well.

The following patient appeared to show an excellent response to the combination of thyroid extract and lipotropic factors, after failing to recover during more than 100 days of thyroid therapy without lipotropic factors. He illustrates also how a relapse may be erroneously attributed to trauma (in his case extraction of impacted molars).

The patient, X.I., was born in 1931. As a child, he was undersized and was considered slow to learn. However, he was able to graduate in the middle of his class from an urban public high school where the faculty considered him an average student. He was rejected for military service at age 20 because of hypertension.

In February 1952, the patient developed a severe upper respiratory tract infection. After 3 days of fever, be became overtalkative, confused and delusional. He was taken to a public psychiatric hospital, where he remained for about 7 months. A pneumo-encephalogram revealed nothing of interest.

In the public hospital, the patient ran a very stormy course. He was confused, delusional and combative much of the time, and often developed a fever of 105°F, which could be reduced easily by electroshock. The patient was given a total of 75 electroshock treatments, without lasting benefit. He was given insulin shock therapy for a brief period, but developed episodic hypertension (blood pressure about 200/110) during his insulin stupors, and sometimes was awakened with difficulty. Laboratory studies ruled out pheochromocytoma.

On admission to the Milwaukee Sanitarium, the patient was very vague, preoccupied, delusional and hallucinated. At times he was combative and required restraint. Physical examination revealed acne and a blood pressure of 145/90.

The blood cholesterol on admission was 262 milligrams per cent. The BMR, during a period of some excitement, was minus 2. Liver function (bromsulphalein retention, cephalin flocculation) and glucose tolerance tests revealed nothing of interest. A normal eosinopenia developed in response to epinephrine. Blood sodium and potassium concentrations were normal. An endocrinologist called in consultation found no evidence of adrenal cortex or thyroid dysfunction.

The diagnosis of periodic relapsing catatonia was not considered at first because no history of improvement and relapse had been obtained from the public hospital. The prognosis was considered to be extremely poor, and it was believed that nothing could be lost by giving the patient thyroid extract. Electroshock was not given because of the large number of such treatments already administered.

The initial response to thyroid extract, vitamins and insulin shock therapy was very gratifying. The patient improved rapidly, developed no increase in blood pressure, and after 34 days appeared to be well. Impacted molars were removed on the 34th day. After 48 hours he relapsed into stupor.

In retrospect, this development was sufficient for the diagnosis of periodic relapsing catatonia. At the time, however, the relapse was attributed to the extraction of the teeth.

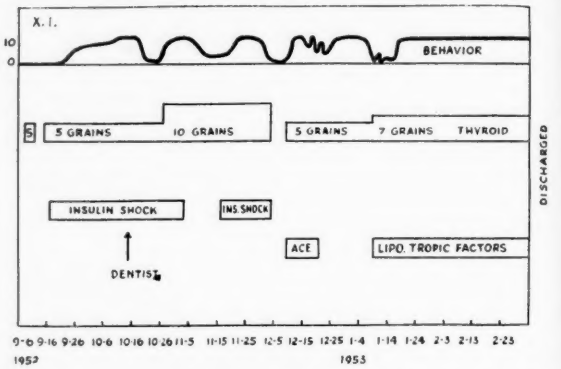

Figure 3. Response of patient X.I. to various types of treatment. The first relapse was preceded by removal of impacted molars; this event is indicated by the arrow and the word "dentist." "ACE" refe;rs to the administration of aqueous extract of adrenal cortex.

The patient's subsequent treatment and course are shown in Figure 3. The apparent response to lipotropic factors (choline, methionine and inositol) in January 1953, is striking. There was no apparent response to injections of adrenal cortex extract, given in combination with oral potassium chloride.

Since his discharge from the sanitarium, February 28, 1953, the patient has been maintained on 7 grains of thyroid extract daily, with vitamins and lipotropic factors. He has resumed his work, and appears to be entirely well.

The next patient illustrates the danger of reducing the daily intake of thyroid extract or thyroxin too hastily in the presence of supposed symptoms of over-dosage.

The patient, T.D., was born in 1928. She appeared to be well until late in 1945 when, following an unexplained fever of 104°F, she developed a manic-like excitement. After 25 electroshock treatments and some insulin shock therapy, given elsewhere, the patient was greatly improved, but she continued to be mildly unstable during 1946. In 1947 she had a total of 12 brief periods of excitement, each lasting about a week, during which the patient became noisy and combative.

She was admitted to the Milwaukee Sanitarium in April 1948. After a very stormy course, she was stabilized on vitamins and thyroid extract with a daily dose of 16 grains of thyroid extract and was discharged in October 1948. The referring physician was very cooperative, and eventually increased the dose of thyroid extract to 20 grains daily.

The patient returned for a routine check-up six months after her discharge. She was free of symptoms, but her pulse rate was 140 per minute. The dose was then reduced to 5 grains daily, in April 1949. The BMR fell very slowly and irregularly to minus 2 in September, 1949, and rose to plus 8 in January 1950.

About Christmas, 1949, the patient began to relapse, and had to be readmitted to the sanitarium. She responded quickly to four electroshock treatments and then was placed on 3 milligrams of thyroxin orally daily.

She remained well through the remainder of 1950 and during 1951. Because of persistent ankle edema, which was present also before treatment, the dose of thyroxin was reduced to 2 milligrams daily in December 1950. A brief relapse occurred in April 1952; this was treated in another hospital, and the dose of thyroxin was reduced to one milligram daily. The patient felt only fairly well in the remainder of 1952, and in November had to be readmitted to the sanitarium because of depression. The dose of thyroxin was increased to three milligrams daily with good effect. The patient has been well again since early in December 1952.

Summary

The results of 44 attempts to treat periodic relapsing catatonia, occurring in 31 patients, have been described. For success, we have found it necessary to use doses of thyroid extract or thyroxin which are larger than those commonly employed by internists, and to use them persistently. Many patients develop temporary side effects which may be alarming, and many patients relapse several times after treatment is started. In spite of these diffiiulties, the results in patients treated for adequate lengths of time and given vitamins with sufficient amounts of thyroid extract or thyroxin are very good.

Shock therapy has been used in some of the cases, without apparent ill effect. The effect of adrenal cortex extract and lipotropic factors, combined with thyroid therapy, should be studied further.

Some statistics on the patients and their treatment, and some illustrative case histories are presented.

Our clinical observations have been in qualitative agreement with a mathematical theory of periodic relapsing catatonia.

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D-Tubocurarine and Flaxedil in Electric Shock

L. G. MCKEEVER, M.D.
Oakland, Calif.

Since Bennett[1] first introduced curare for the prevention of traumatic complications in convulsive shock treatment, a number of other medications and techniques of treatment have been proposed for the same purpose. Substitutes such as beta erythroidine, magnesium sulfate, quinine methochloride, Myanesin, Seconal, Pentothal, decamethonium bromide, and Anectine have differed from d-tubocurarine in their mode of action.

Tri (diethylaminoethoxy) benzene triethyliodide, synthesized in France and introduced under the name Flaxedil, was reported by Bovet, Depierre and de Lestrange[4] to have pharmacological effects and mode of action almost identical to that of d-tubocurarine. An additional mild vagolytic action has been demonstrated by Wescoe and Riker[11] who report slight increases in blood pressure and heart rate not affected by strong vagal stimulation. Flaxedil is antagonized by prostigmine and Tensilon. It is reported to have shorter duration of action than d-turbocurarine.

Several reports appeared in the foreign literature[7,8,9] regarding the use of Flaxedil in electric shock treatment, and recently Holt, Niswander, Ziegler and Turk[6] have reported its use compared to Syncurine for maximal modification of convulsions in patients for whom electric shock treatment would otherwise have been contraindicated.

The purpose of this study is to compare Flaxedil with d-tubocurarine for routine use in the prevention of traumatic complications, principally in patients with normal risks, and particularly with reference to the determination of optimal dosage.

The method was the use of Flaxedil and d-tubocurarine interchangeably in the manner which had been previously established for d-tubocurarine. Usually the first treatment was given with d-tubocurarine, with the dose calculated as described by Bennett and averaging one mg. per 13 pounds of body weight. Sodium pentothal, 2½% solution, was given in amount necessary to allay pretreatment anxiety. The two medications were given independently by use of a three-way stopcock. If the first treatment was followed by excessive salivation to the point where the airway was in the least obstructed, 0.6 mg. of atropine was added to the curarizing solution for subsequent treatments. Pentothal was given slowly to the point where the patient lost ability to count; the relaxant, sometimes with atropine, was given in an injection of one minute duration; the needle remained in place during a waiting time of three minutes during which more Pentothal was given if patient became restless. Removal of the needle was followed by the electric stimulation. Oxygen was administered before or after treatment by a mask with rebreathing bag if even slight cyanosis or use of accessory muscles of respiration appeared before treatment, or if post-treatment cyanosis lasted for more than a few respirations. Guedel airway and pharmacologic antagonists were always in the treatment room. Patients were not held down or hyperextended. Treatments were given by the author, usually with one experienced nurse assistant. An additional assistant was obtained whenever possible for the first treatment of each patient. The limitations imposed by private practice, personnel, and Pentothal prevented the production of a completely controlled investigational study.

It is to be pointed out that in this series only partial curarization was desired. The goal, with the exception of three patients who had previous fractures was a level of curarization which would result in a convulsion previously described by Bennett, McKeever and Turk[3] as "two plus" in strength. This level of relaxation which was proposed as optimal for prevention of traumatic com-

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plications in "normal risk" patients was described as follows: "The patient can raise the arm but not the head; grip strength as measured by dynamometer decreases 85 to 95 per cent under pretreatment performance; the ensuing convulsion is so modified as to eliminate outcry, opisthotonus, or raising the legs from the bed; and throughout the clonic phase the forearms can be easily extended by the observer when held only between the thumb and one or two fingers." Further, at this level of curarization spontaneous respiration is maintained before treatment at a sufficient volume that, except in old or emphysematous patients, no cyanosis appears and accessory muscles of respiration are not called into play. The first post-treatment respiratory efforts are vigorous, and post-treatment cyanosis is cleared by the first four or five respirations. Of course this ideal degree of relaxation was not always achieved for the first treatment.

According to this method 1093 electric shock treatments were given to 72 patients. 580 treatments were given with Flaxedil; 513 with d-tubocurarine. Nearly all of the patients received both drugs, but not necessarily alternately or for the same number of treatments. Early in the series the tendency was to use d-tubocurarine more frequently, especially if there were any difficulty in adjusting dosage or any increased risk was anticipated. Later, as confidence was established, Flaxedil was given more often than d-tubocurarine. This series includes all the private patients treated with electric shock during the period of the investigation. Thirty of the 72 patients were given all or part of the treatments while hospitalized; others were outpatients. Two types of electric shock were given: Conventional Cerletti-Bini, and brief stimulus treatment. In general conventional treatment was used for more disturbed patients who were hospitalized, and brief stimulus treatment was used for those less severely ill who were handled as out-patients. The patients were given from one to 57 treatments. The age range was 16 to 83. Eight patients were over 70 years of age. The clinical conditions treated were predominately depressions, with several acute schizophrenic reactions, and a few manic-depressive, manic psychoses. Three patients had previously suffered compression fractures sufficiently severe to interrupt treatment elsewhere.

One of the first problems to be resolved was determination of the dose of Flaxedil necessary to produce the desired "two plus" convulsion. Since Unna, et al.[10] had reported that 48.53 mg. of Flaxedil equalled 9.45 mg. of tubocurarine chloride in producing 95 per cent decrease in grip strength, the first approximation of comparable dosage was made on the basis of a 5 to 1 ratio. Since this did not produce enough relaxation with Flaxedil, the ratio was increased to 6 mg. of Flaxedil to 1 mg. of d-tubocurarine. To establish this ratio exactly, a comparison of dosage was made on the first 27 patients with whom "two plus" strength convulsions were obtained while using both drugs. This group illustrates not only the ratio of doses, but also the total dose of these medications necessary to produce modification to "two plus" convulsions. Among those 27 patients, however, there were no large muscular men, and there were several small women. The dosage range of Flaxedil was 40 mg. (2.0 cc) to 84 mg. (4.2 cc.), the mean dosage being 62.2 mg. (3.1 cc). The range for d-tubocurarine was 6.6 mg. (2.2 cc.) to 13.5 mg. (4.5 cc.) with a mean dosage of 10.14 mg. (3.4 cc.). Dividing 62.2 by 10.14 gave a final observed ratio of 6.13 mg. of Flaxedil to 1 mg. of d-tubocurarine. Since this difference from the 6 to 1 ratio was too small to be clinically significant, the simpler ratio was continued for determining equivalent dosage. To reduce this to volume of solution as dispensed, 0.9 cc. of Flaxedil (20 mg./cc. solution) was found to be equivalent to 1.0 cc. of d-tubocurarine (3 mg./cc. solution).

Although the average dosage for "two plus" convulsion with Flaxedil was found to be 1 mg. per kg., correlating with the average dose for d-tubocurarine of 1 mg. per 13.3 pounds, the same factors were found to control individual variation from this dosage as with the natural curare. For example, in one heavily muscled 154 pound (70 kg.) man the "average dose" of Flaxedil would have been 70 mg., but 80 mg. was actually necessary to reduce the convulsion to "two plus" strength. The "average dose" of d-tubocurarine would have been 11.6 mg., but 13.5

mg. was necessary. A rather obese, small muscled woman of the same weight was sufficiently relaxed with 64 mg. of Flaxedil or 10.2 mg. of d-tubocurarine.

In confirmation of the "fatigue factor" reported by Bennett[2,3] for d-tubocurarine and by Holt, et al.[6] for Flaxedil, which sharply increases the effect in the presence of fatigue, agitation, overactivity and exhaustion, a woman patient weighing 124 pounds (56 kg.) responded when calm with a "two plus" convulsion to the "average" 9.6 mg. of d-tubocurarine or 56 mg. of Flaxedil; but when agitated and resistive she was relaxed to a greater degree with only 6.6 mg. of d-tubocurarine or 40 mg. of Flaxedil. Her resistance and agitation apparently increased the effects of both medications by 50 per cent.

No exception was observed to the expectation that there would be a perfect correlation of the doses in all patients. However, the methods of this study were not sufficiently exact or well enough controlled to establish proof of this correlation.

The largest dose of Flaxedil used was 140 mg. The patient was a 170 pound, lean, muscular, 52 year old man, manic-depressive, depressed. A history was obtained of two previous compression fractures, one several years previously sustained during hazing stunts when crossing the equator, and the second a few months previous to our treatment when he received an electric shock treatment with curare at a Veteran's Hospital. Records from the hospital did not reveal the dose or exact preparation given. X-ray revealed a generalized osteoporosis of moderate degree and wedging of two thoracic vertebrae. Since greater than usual relaxation was sought the first dose was 100 mg. instead of the calculated 77 mg. Preparations were made for immediate oxygen insufflation and administration of antagonists. A convulsion of "two plus" strength was obtained, which we deemed too severe for complete safety in this patient. The dose was increased 10 mg. with each treatment until the optimum dosage of 140 mg. was reached. At this dose spontaneous respiration was maintained, but tidal exchange was small, so that oxygen was administered from the time Flaxedil was given until the treatment. A very mild convulsion was produced during which the arms remained extended. The needle was kept in the vein during treatment so that Tensilon, 20 mgm. could be given immediately after the end of the convulsion and oxygen was continued for 20 to 30 seconds until respirations were strong. The degree of relaxation in this case was less than described by Holt, et al.,[6] as their routine, but was sufficient to prevent damage in the presence of osteoporosis.

We encountered only one case in which an unfavorable reaction occurred when Flaxedil was used. An obese, 60 year old, depressed white female, hospitalized at a sanitarium where curarization is rarely performed, had been given one treatment satisfactorily with 10 mg. of d-tubocurarine, one with 58 mg. of Flaxedil, and one with 60 mg. of Flaxedil, which gave more relaxation than deemed necessary. On the fourth treatment, the usual 200 mgm. of Pentothal was given followed by 56 mgm. of Flaxedil, and the needle was removed. The treatment apparatus was being connected and electrodes attached, with some distraction of attention from the patient. Two minutes after injection of Flaxedil was completed it was noticed that patient's respiration was obstructed by the tongue having fallen against the palate. There was no tidal exchange, although there were movements of both diaphragmatic and intercostal muscles. The patient was rapidly becoming cyanotic. Jaws were clamped shut tightly, indicating that the patient was not overcurarized. During the minute or two that were required to force in a Guedel airway, obtain oxygen from another room, and perform another venipuncture, the cyanosis became very deep, respiratory efforts ceased, and pulse became weak and rapid. The patient responded after prostigmine i.v., adrenalin i.m., oxygen inhalation and artificial respiration. This dangerous reaction was not attributable to Flaxedil, but to several deficiencies in treatment routine. (1) The i.v. needle was removed immediately after Flaxedil was given, so that time was spent in another venipuncture for the antagonist. (2) Patient was not carefully observed for onset of respiratory obstruction, since attention was being given to the treatment apparatus. (3) Oxygen was not in treatment

room and an adequate mask and bag was not available for insuflation. This dangerous reaction was not due to overcurarization, but rather to under oxygenation.

The final results of observation of the reaction of these 72 patients, receiving 1093 electric shock treatments, was that there is no significant difference in the two medications, as they were used in moderate amount in this clinical situation. The only observable difference in this series was a slightly more rapid recovery rate with Flaxedil than with d-tubocurarine. This difference was not great. No attempt was made at an objective measurement. It was apparent only with patients treated with brief stimulus therapy, who regained consciousness more quickly, and who complained somewhat less of a feeling of weakness when they received Flaxedil. This difference was small. Many patients, even on questioning, could not report any difference in this respect. In this series, there was no death, and no clinical evidence of either new fractures or aggravation of previous fractures. One unfavorable reaction to premedication led to the omission of one treatment.

The effect of Flaxedil, like that of d-tubocurarine, was found to be effectively and quickly antagonized by both prostigmine and Tensilon. In the patients where great relaxation was desired oxygen was given, and one of the antagonists was given routinely after treatment. In some patients who complained of weakness after treatment the antagonists were also given. The usual dose of Tensilon used was 10 mg.; of prostigmine it was 0.5 mg.

Discussion

Muscle relaxants are used in connection with electric shock treatment for two purposes, although there is no definite line of demarcation between these two uses. First, as originally proposed by Bennett,[1] relaxants may be used routinely in all patients to prevent traumatic complications. For this purpose, safety, moderate, predictable, and controllable degree of relaxation, simple technique, requiring neither a great deal of time, nor extra personnel is sought.

Secondly, relaxants may be used, as recently described by Holt, et al.,[6] and Gottlieb and Morris,[5] to make possible treatment in patients who otherwise would not be treatable because of recent fractures, osteoporosis, cardiac disease, or other conditions which contraindicate even mild convulsions. For this purpose, since the risks of no treatment or of unmodified treatment are great, the curarization may legitimately carry some risk. A great deal of relaxation is usually desired; more complicated techniques, requiring extra time and personnel are acceptable, although not desirable; predicability of partial relaxation is not so necessary, since almost total relaxation is desired. Few patients require this type of management.

This paper deals principally with the routine use for prevention of trauma in patients who are normal risks. In only three of the patients treated in this series, the ones with previous fractures, was greater than moderate relaxation desired, and in only one (the case cited) was anything approaching complete relaxation attempted. The average dose in an unselected group of these patients who attained similar moderate relaxation on both drugs was 62.2 mgm. of Flaxedil and 10.1 mg. of d-tubocurarine. The patient cited in whom great relaxation was achieved required 140 mgm. of Flaxedil, which is comparable to the dosage range of 100 to 180 mgm. reported by Holt et al.,[6] for "maximal softening of the seizure with Flaxedil." Similarly, "complete curarization" with d-tubocurarine was attained by Gottlieb and Morris[5] with doses ranging from 21 to 52 milligrams, which is two to five times the average dose of 10.1 mgm. with which partial curarization was produced in this series. This great difference in dosage makes necessary a fundamental difference in technique. With partial curarization for routine use the dose is calculated to be well under the amount which produces apnea. With "complete curarization," apnea and the possible need for artificial ventilation of the patient is to be expected. Consequently, the personnel and equipment must be available for the apneic patient—particularly someone who is adequately trained in oxygen insufflation and is equipped for tracheal intubation if necessary.

The large doses used in complete curarization with positive pressure breathing in pa-

tients with cardio-vascular and other lesions is convincing evidence of the great margin of safety when these substances are used for partial curarization *if adequate oxygenation is maintained.* As illustrated by the one unfavorable reaction described, asphyxia will result in apnea when the dose of curare is well below that at which apnea would ordinarily occur. As Bennett[2] has pointed out, "the sure method of resuscitation to save such a patient before asphyxia causes cardiac failure is insufflation with oxygen." I would propose going a step farther and recommend the prevention of even mild degree of asphyxia by early use of oxygen at the first sign of cyanosis, use of accessory muscles of respiration, respiratory obstruction, or greatly reduced tidal exchange. Early oxygen administration prevents gasps for air, which cause the jaw to drop and allow the tongue to fall against the palate with resultant obstruction and stridor. Mild positive pressure prevents the relaxed vocal cords being sucked together like a valve closing.

In summary, observations of 580 electric shock treatments before which Flaxedil was routinely administered to prevent traumatic complications, compared with 513 treatments in which d-tubocurarine was administered, leads to the conclusion that the action of Flaxedil is almost identical to that of d-tubocurarine.

It was found that the effect of a calculated dose of Flaxedil was as predictable as that of d-tubocurarine, and that the same factors (body weight, "muscularity," degree of fatigue) affected the dosage.

No unsatisfactory side effects were observed; effective antagonists are available; as with d-tubocurarine, myasthenia gravis is an absolute contraindication. Since Flaxedil is a relatively new medication, the safeguards necessary in its use are less well established than for d-tubocurarine. It is concluded that Flaxedil is a satisfactory product for routine use in the prevention of traumatic complications of electric shock treatment.

Author's Note: Flaxedil used in this study was supplied by Lederle Laboratories, Pearl River, N. Y. D-tubocurarine was supplied by Endo Products, Inc., Richmond Hill, N. Y.

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Sodium Amytal Rorschach in Incipient Schizophrenia

MARVIN ZUCKERMAN, SANFORD IZNER, M.D., and RUDOLF LEISER, M.D.
Eloise, Michigan

The clinical psychologist is frequently confronted with patients who are too withdrawn or rigid to give definitive data for a psychological appraisal. In these cases a Rorschach given while the patient is under the influence of sodium amytal is often helpful in clarifying the diagnosis. Kelley and Levine[1] found that diagnoses made from amytal Rorschachs tended to agree with clinical diagnoses in a large proportion of cases. These authors state:

"As can be seen . . . diagnosis was facilitated by both the quantitative and qualitative changes, and apparently the amytal itself produced no other change in the patient's Rorschach responses than to permit a free flow of basic responses" (p. 72).

Layman[2] tested schizophrenic patients before, during and after amytal administration. He found significant increases in the score of organizing ability and in the number of responses given in the amytal Rorschach. Whereas the organization score dropped in the post-amytal Rorschach the number of responses remained at the increased level.

The amytal technique used in this case was as follows: A freshly prepared 10 per cent solution of sodium amytal was injected intravenously at the rate of approximately one c.c. per minute. The injection was continued until the patient became drowsy. The proper level of sedation was determined by having the patient count from 1 to 20 forward and backward. The injection was stopped when the patient began to make errors.

According to Noyes,[3] "In this lowered state of consciousness midway between being awake and being asleep, inhibitory processes are released, rapport is produced, and suppressed and conflictual material is brought into consciousness" (p. 121).

The following case is presented as a striking illustration of the usefulness of the sodium amytal Rorschach in clarifying diagnosis.

From Department of Psychiatry, Wayne County General Hospital.

Social History

The patient, D.B., is a 28 year old, divorced, white, Protestant male. He was the youngest of five siblings. The mother had a difficult time with his birth and instruments were used. He experienced convulsions when teething at the ages of one and seven. He was described as a demanding child who was never able to form a warm relationship with his father but became attached to other members of the family such as his grandmother and sister. His father would make decisions for him and he would comply with his father's wishes without complaint. Two siblings were adopted into the home, one when the patient was 10, the other when he was 14. The second time he became quite aggressive toward the younger, adopted child. This was unlike his previous general behavior which was described as "timid." When his grandmother died he felt a great loss and seemed to lose interest in his environment. After a few months he recovered from this reaction. His father made him leave school after the ninth grade and he began to work as a house painter with his father.

Prior to his entry into the Army in 1942, D.B. rarely sought female companionship. He did have many male friends with whom he visited taverns. He was in the Army for four years and was wounded slightly in the Battle of the Bulge. After his discharge he bought a farm with his father. He could not make a living on the farm and went to live with his sister feeling very guilty about leaving his parents on the farm. In the summer of 1950 his parents introduced D.B. to an attractive 19 year old girl whom they had known for several years. He married this girl who turned out to be pregnant at the time he married her. He later found out that his best friend was responsible for her pregnancy. His wife continued to date other men and six weeks after their marriage she deserted him. Soon afterwards D.B. began to develop symptoms of withdrawal, anxiety, and depression.

Psychiatric Observations

The patient was admitted on 10-5-51. On 10-8-51 he was noted to be slow in responding but cooperative and coherent. He expressed many feelings of inadequacy. He stated he was "scared." He would sit in a catatonic fashion, chewing his thumbnail and responding slowly and abruptly to questions. He was able to describe the circumstances leading to his admission and stated, "I just didn't have a mind of my own. I got so used to working for my father. I got a job with a lot of responsibility. . . . I wasn't used to it and the responsibility was too much and I was afraid of the job so I just let go." He related how he became depressed over the failure of the farm and his marriage and his inability to meet the high cultural standards in his sister's home. He said he was always shy and rarely went out with girls. He expressed considerable ambivalence toward his parents especially with regard to his father. He was emotionally appropriate during the entire examination. No ideas of unreality or hallucinatory experiences were elicited. He expressed many nihilistic ideas, among others the feeling that his body was wasting away and that he was afraid he would die. Physical findings were all within normal limits.

The psychiatric impression at this time was: Psychoneurosis, mixed type, severe, manifested by depression and anxiety. At the same time the psychiatrist felt that the patient was emotionally appropriate, that his judgment was unimpaired and that his rapport was fairly adequate. The patient's somatic preoccupations or delusions were strongly suggestive of an early schizophrenic process.

Psychological Testing

When first seen on 10-8-51 the patient was in an anxious and depressed state. As the testing progressed he became slightly more relaxed but was unable to concentrate sufficiently to take the intelligence test. In the middle of a test he began talking about his illness with a great deal of pessimism, believing he was near death. He would also reiterate several thoughts, e.g., "I wasn't able to think for myself, that's my trouble." He said that his father did all his thinking for him. He also blamed his condition on an "inferiority complex" which he said stemmed from the fact that he was hard of hearing.

On the first administration of the Rorschach (see table 1) the patient responded to the whole impression of every card with the response "bones." His only other re-

TABLE 1
Rorschach profile: 1st test, 10-8-51

W	10	F+	0	At	10	*R-T achromatic*		*R-T colored*		av. R-T	41.6"
D	1	F—	10	Bld	1	I	140"	II	80"	T/R	46.8"
		M	0			IV	20"	III	50"		
R	11	FM	0	P	0	V	15"	VIII	32"		
		C	1			VI	14"	IX	20"		
						VII	14"	X	30"		
W%	91%	F%	91%								
D%	9%	F+%	0%								
		M:C	0:1.5								

Note:

1. The lack of intellectual control reflected in the poor form quality of the responses (F+%).
2. The lack of ability to differentiate or analyze stimulus impressions (ahigh W% composed of responses of poor quality to whole stimuli).
3. Anxiety and inhibition as revealed in:
   a. Underproductiveness or a small number of responses (R).
   b. Long reaction times to stimuli (av. R-T) and especially long reaction times to the colored or emotional stimuli (R-T colored).
   c. Long periods of time spent on each response (T/R).
   d. Lack of ability to express fantasy (M and FM) and the large percentage of rigid form responses (F%).
4. The indication of a possible disturbed emotional life underlying the patient's emotional inhibition. Note the one uncontrolled color response (C).
5. The preoccupation with the somatic condition reflected in the large number of anatomy responses (At).

sponse was the reaction "blood" to the top red detail of card II. The perseverated anatomy response was interpreted to mean that D.B. was so preoccupied with his somatic condition that he could not attend to his environment with any degree of intellectual control. This would explain his inability to take the intelligence test. The one pure color response was taken to mean that his emotional life might also be uncontrolled. However such an unproductive Rorschach is of dubious validity and tells us little beyond the fact that anxiety and depression have severely inhibited the patient's functioning. His reaction times, listed in table 1, show a great amount of inhibition on the initial test stimulus and subsequent inhibition on every colored test stimulus. This "color shock" is an indication of severe anxiety in emotional, interpersonal relationships.

Because of the patient's underproductiveness the examiner began to test the limits of his perceptual ability. After a few responses were suggested to the patient, and accepted by him, he began to point out spontaneously common, differentiated percepts. At the time, this was taken to indicate that with a little social stimulation D.B. was able to deal with reality in a nonpathological manner. As will be seen, however, this method known as "testing the limits" was not predictive of the course of the illness.

This patient was given the Rapaport Word Association Test and responded in a neurotic and depressed manner with many long reaction times and a moderate number of association disturbances. To the word "love" he responded "mother," but he blocked on the word "father" and commented, "It makes me think too much of the past." It took him 20 seconds to respond to the word "suicide" and then he replied "myself." Suicide was noted to be a threat in this case by both the psychologist and psychiatrist. (Subsequently a suicidal attempt was made.)

On the Thematic Apperception Test the patient was unable to maintain any ego distance from his stories. His stories actually consisted of brief personal associations to the pictures. He saw a mother figure and hero figure mourning over the death of a father figure and commented spontaneously: "My father is sick now . . . I wouldn't be a bit surprised . . ." He seems to regret this death-wish on the next card where he commented on a father and son picture, "Father knows best." His ambivalence toward the father was abviously a great source of conflict. He looked at a picture which is often interpreted as an older woman choking a younger one and said: "She's giving her affection. I never got much affection." He said that his parents were too old-fashioned to be affectionate. Apparently he felt rejected by the mother figure and tried to deny any implications of hostility toward her.

The patient's Bender-Gestalt reproductions and figure drawing test indicated the degree to which the patient's mental energy was blocked. His Gestalt figures were small and sketchy and when he began a series of dots they tapered off after a brief attempt to reproduce the entire series. His drawing reflected extreme rigidity and a depersonalization trend. The hand and crotch regions of the figure were missing which probably indicated his feelings of inadequacy in dealing with his environment and his feeling of sexual insufficiency.

No positive diagnostic conclusions were drawn from this testing. The possibilities included a severe neurasthenia and an incipient schizophrenia. His ability to recover when "testing the limits" on the Rorschach seemed a hopeful sign. A Sodium Amytal Rorschach was suggested in order to clarify the diagnosis.

The Sodium Amytal Rorschach

The second Rorschach was given on 10-9-51 with the patient under the influence of Sodium Amytal. At the beginning of the injection he was extremely agitated and resistive. A large dose (approximately 7½ grains) of amytal was necessary before he became relaxed. At this point he sat up smiling for the first time since his admission.

The main change in the amytal Rorschach (table 2) was the loss of inhibition which characterized his previous Rorschach. He became more productive offering 25 responses. He took less time in responding to the stimuli and gave his responses with greater rapidity. The perseveration of the response "bones" to all stimuli on the previous Rorschach had indicated intellectual inadequacy. Now this response was given

TABLE 2
Rorschach profile: 2nd test, 10-9-51,
Sodium Amytal

W	3	F+	12	H	3	P 7	*R-T achromatic*		*R-T colored*		av. R-T 6.5"
D	21	F—	2	Hd	1		I	5"	II	3"	T/R 20 5"
d	1	M	4	A	4		IV	15"	III	3"	
		FM	1	Ad	4		V	3"	VIII	3"	
R	25	c'	1	Aobj	1		VI	4"	IX	20"	
		C	5	Bld	6		VII	1"	X	2"	
				Cg	2						
W%	12%	F%	56%	Obj	2						
D%	84%	F+%	83%	Ls	1						
d%	4%										
		A%	32%	M:C	4:7.5						
		P%	28%								

Note the following changes from the first test (table 1):

1. Gain in intellectual control (increase in F+%).
2. Increased ability to differentiate and react to parts of the whole stimulus (decrease of W%, increase of D%).
3. Reduction of anxiety and inhibition.
   a. Increase in productivenes (R).
   b. Shorter reaction times (av. R-T).
   c. Faster flow of responses (T/).
   d. Increase in fantasy responses (M and FM) and decrease in rigid form responses (F%).
4. Loss of inhibition in responding to emotional stimuli (R-T colored) and the free expression of uncntrolled and regressive emotional responses (increase in C).
5. Diversion from the preoccupation with the somatic condition (decrease in At).

only once. However, a more pathognomic change appeared in his response to emotional stimuli (color) while under amytal.

He had previously given one uncontrolled emotional response but had reacted with inhibition when confronted with the emotional stimulus of color. With the disappearance of this neurotic type of "shock" reaction he showed the full-blown expression of a tendency to impulsive, regressed emotional reactions. Whereas he previously had given one "blood" response, he now began to see blood in almost any red area before him and he even saw blood in one black area. Previously he had perseverated an inappropriate intellectual response ("bones"). Under amytal, he perseverated an inappropriate emotional response. The amytal Rorschach seemed to indicate that when his defenses were lowered the patient reacted with the underlying emotional reactions of schizophrenia. The patient was therefore described as an incipient schizophrenic.

Under amytal the patient revealed anal and homosexual contents in his fantasy. On card III he saw men going to the toilet but tried to deny the self-reference of this response by making the men cartoon characters. On card IX he saw men looking and pointing at each other indicating a suspicious attitude toward others which could develop into a paranoid trend. On card VII he saw women wanting to kiss, indicating his need for love.

On the testing of the limits of the amytal Rorschach the patient tried to mobilize his defenses again. Instead of the uncontrolled reaction to color he tried to inject some structured form elements into those areas. The men seen as going to the toilet became men "getting married." When pressed too hard he tended to confabulate. Pressed as to why the men were pointing at each other, he finally tried to escape by saying that they were not pointing at each other but were pointing at a "crab's leg," an irrelevant detail he saw between the two areas he was speaking of as men.

After the amytal Rorschach, the psychiatrist questioned the patient about his difficulty with his wife. The patient smilingly said he was "like a jack-rabbit" referring to his ejaculatio praecox tendency. He was apparently unable to satisfy his wife, contributing to her infidelity.

Clinical Process

By 11-7-51 the patient showed considerable improvement. He was more friendly and talked freely to other patients and to employees. He ate and slept well. At this

date he was receiving electric shock treatments.

On 11-11-51 D.B. was noted to be somewhat disturbed and was talking continually.

On 11-12-51 the patient broke down and demonstrated hebephrenic-like behavior. The entry in his chart reads as follows:

"The patient is reported to be quite disturbed. He was seen eating feces yesterday and had to be restrained. Patient's affect is quite flat today. He looks puzzled. He states that he had 'a funny feeling' in his legs but does not elaborate on this. He also complains of pressure in his rectum. He is seen sitting with his finger in his rectum 'to relieve pressure.' Patient shows definite somatic delusions and a marked change from several days ago."

Final Testing

The patient was retested on 11-13-51 without the use of amytal. This Rorschach (table 3) revealed a combination of the lack of intellectual control demonstrated on his first Rorschach and the impulsive, regressed affectivity demonstrated on his amytal Rorschach. His productivity and reaction times indicate that the loss of inhibition on the amytal Rorschach was still in effect. The main difference between this test and the amytal test was the loss of intellectual control because of the perseveration of the vague anatomy response. Under amytal the patient was able to turn away from this unrealistic somatic preoccupation. Now it had returned together with the uncontrolled affect in the form of a complete psychotic breakdown.

In his figure drawings we also found deteriorative change. His figures were drawn sideways as if to illustrate the turning away from reality. The body outlines had become vague, reflecting the loss of ego-boundaries. This time the female figure was drawn first and larger, indicating the increase in strength of his homosexual-anal component. The inside of the male figure was devoid of features and the figure lacked arms and legs, reflecting his depersonalization and complete loss of adequacy. The drawings seemed to parallel the clinical change in the patient since the first testing shortly after admission.

The change in his Bender-Gestalt test was interesting. He arranged his drawings in succession up one edge of the paper as if trying to cling to his environment for support.

In summary of the last testing, D.B. was found to be in a state of emotional and intellectual regression. The ego boundaries with the environment were vague and his inner feeling evidenced loss of adequacy and identity and a turning away from reality.

Subsequent Clinical Progress

On 11-20-51 the patient attempted suicide by hanging himself with a belt. He was discovered before he could hurt himself.

By 12-4-51 the patient had demonstrated

TABLE 3

Rorschach profile: 3rd test, 11-13-51, after psychotic breakdown

W	7	F+	6	H	3	P 8	*R-T achromatic*		*R-T colored*		av. R-T 4.8"
D	18	F—	9	Hd	1		I	13"	II	4"	T/R 18.5"
S	1	M	3	A	4		IV	2"	III	4"	
		FM	3	Ad	4		V	4"	VIII	3"	
		CF	1	Aobj	1		VI	5"	IX	4"	
		C	4	At	1		VII	7"	X	2"	
				Bld	6						
W%	27%	F%	58%	Cg	2						
D%	69%	F+%	40%	Obj	2						
S%	4%	A%	23%	Ls	1						
		P%	31%								
		M:C	3:7								

Note the following changes from the second test (table 2):

1. Loss of intellectual control previously demonstrated (F+%).
2. Return of somatic preoccupation (At).
3. Stabilization of loss of inhibition and regressive emotional tendency revealed under amytal (C).

a steady improvement. He was pleasant, cooperative, mingled with the other patients and was eating well.

On 12-21-51 the patient went home for a Christmas visit. It was subsequently discovered that his parents had taken him to a minister who gave him a good "talking-to" and had also taken him to a dance.

On 12-26-51 D.B. returned from his visit confused, upset, and not eating well.

On 12-28-51 D.B. was started on a second series of EST; the first series of 17 shocks having been completed 12-12-51.

Final Psychiatric Diagnosis

The psychiatrist's diagnostic impression at the time of admission was severe psychoneurosis with possible early schizophrenic process. The psychiatrist is now of the opinion that the patient had experienced a breakdown of his neurotic defenses while in the hospital and that he is now a schizophrenic with mixed catatonic and paranoid features.

Discussion and Summary

The features of this case are not presented as scientific proof of the efficacy of Sodium Amytal but merely as a case illustration of these effects. Certainly no generalization should be drawn from this single case but it may be valuable as a guide to research in this interesting area.

This case offers support for the Noyes statement that Sodium Amytal releases inhibitions and that suppressed and conflictual material is brought into consciousness. The inconclusive, rigid approach of the first Rorschach changed to a free-flowing response to the Rorschach under amytal. The emotional tendencies, which previously produced anxiety reactions exploded in full strength under amytal. The anal fantasy revealed under amytal may be compared with the patient's very meticulous toilet behavior before his breakdown and his regressive soiling behavior after the breakdown.

The case supports the findings of Kelley and Levine that the amytal Rorschach clarifies diagnosis. At a time when the patient was manifesting ambiguous defenses and reactions the amytal Rorschach revealed the emotional regressive tendency which was borne out in the patient's behavior after his breakdown. The amytal Rorschach was predictive of the breakdown. The "testing the limits" procedure was not predictive in this respect.

Support is given to Layman's finding that productivity increases in the amytal Rorschach and stays at the raised level after amytal. Beck's "Z" score of organization was not used but it was noted that intellectual control, reflected in the form quality of the responses, increased under amytal and decreased after amytal. It is hard to say whether the results of the post-amytal Rorschach were due to any special effects of amytal or to the sudden exacerbation of the psychosis previous to the last test. It is the opinion of these writers that amytal made the patient more outspoken and reduced his anxiety thereby enabling him to turn his attention away from his somatic preoccupations which were diverting his attention from the world about him.

Rorschach studies performed on normal persons should be done under the influence of amytal in order to determine the general psychological effects of the drug. This interesting technique is recommended in borderline psychotic states and for further follow-up studies to check on the predictive validity of such cases.

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Intraspinal Tumors

(Report of Twelve Cases)

F. KEITH BRADFORD, M.D.
Houston, Texas

An occasional report of patients with tumors of the spinal cord may help to inspire the vigilance necessary to detect these lesions in their relatively early stages. The number of cases reported is small, but each case can be seen to offer its particular problems. The critical degree of spinal cord involvement may appear late in the course of a spinal cord tumor, which, from the earlier symptoms produced, can be judged to have been present for many years. A brief case history of each of the 12 patients is presented with comments on each group. In the series are 4 neurofibromas, 2 meningiomas, 2 lipomas, 1 dermoid cyst, 1 ependymal cyst and 2 osteochondromas.

Case 1. Neurofibroma: S. B. W., a white male aged 37 years at time of laminectomy, September 3, 1947, at Memorial Hospital, gave a history of progressive weakness and numbness in both lower extremities and slight sphincter disturbances for 4 months. Pain radiating from the thoracic spine about the right groin had been present for 5 years. Poliomyelitis at the age of 14 months had left the patient with marked paralysis and atrophy of both lower extremities. Neurological examination revealed marked muscular atrophy with absence of tendon and superficial reflexes. Sensation to pain, temperature, vibration and position was grossly diminished below a line 3 cm. above the groins. Lumbar puncture revealed a complete manometric block. Yellow, clotting cerebrospinal fluid containing 430 mgm. per cent total protein was removed. One cc. of pantopaque was shown by x-ray to be arrested in its upward passage at the 12th thoracic vertebral body. Complete removal of the tumor was readily accomplished and it was shown grossly and microscopically to be a neurofibroma. Motor power rapidly returned to the state left by the poliomyelitis. Follow-up on April 21, 1953, indicates that the patient has had no further difficulty.

Case 2. Neurofibroma: S. C. H., a white male aged 23 years at time of laminectomy, October 26, 1948, at Methodist Hospital, gave a history of a 10 foot fall, astraddle a pipe, on August 16, 1948. Pain and numbness in the right hip, aggravated by coughing or sneezing, and nocturia were the only complaints. The neurological examination was negative except for flattening of the lumbar curve, limitation of back movements and limitation of straight leg raising. The spinal fluid total protein was 371 mgm. per cent. Myelography revealed a block at the 5th lumbar interspace and a filling defect on the right at the 3rd lumbar interspace. A neurofibroma was removed intact at the 5th lumbar interspace with sacrifice of one small nerve filament. At the 3rd interspace a fissure was present in the disc and this disc was enucleated. The patient complained of severe headache and back ache for several months postoperative. Recovery appeared essentially complete when the patient was last examined 13 months postoperative.

Case 3. Neurofibroma: D. L. McL., a white male aged 23 years at time of laminectomy, September 2, 1952, at the V. A. Hospital, Houston, gave a history of bilateral sciatic pain extending as low as the popliteal spaces, which started abruptly 3 months before. The neurological examination was negative except for limitation of back movements and limitation of straight leg raising. Two spinal fluid examinations revealed 140 and 248 mgm. per cent total protein respectively. Myelography revealed a block opposite the 2nd lumbar vertebral body. When the 2nd and part of the 3rd laminae were removed and the dura mater was opened, no tumor was at first evident. However, when a small catheter was passed downward 2.5 cm., a neurofibroma popped upward into the exposed area. Complete removal was effected with sacrifice of one medium sized nerve root. The patient showed no postoperative neurological deficit and made an excellent recovery. Mild back symptoms were present at a follow-up examination, January 22, 1953.

Case 4. Neurofibroma: I. A. J., a white male aged 40 years at the time of two laminectomies, performed October 9, 1952 and December 5, 1952 at Hermann Hospital, gave a history of circling pain in the right upper chest extending to the epigastrium, weakness of the right lower extremity and paresthesias below the 8th thoracic segment on the left of 6 months' duration. Neurological examination revealed spasticity of both lower extremities with weakness more marked on the right, and a partial sensory level at the 8th thoracic dermatome. Lumbar puncture showed no definite block, but the total protein was 110 mgm. per cent. Myelography before the first operation showed free passage of pantopaque into the cervical region. Exploration at the 3rd, 4th and 5th thoracic vertebrae was negative, and a catheter passed freely upward and downward. Myelography two months later revealed a complete block at the lower margin of the 1st tho-

From the Department of Surgery (Neurosurgery), Baylor University College of Medicine.

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racic vertebra. A neurofibroma was removed from beneath the 7th cervical and 1st thoracic laminae to the right of the midline. The postoperative course was uneventful and the patient was ambulatory from the 4th to the 7th postoperative day. On the 7th postoperative day the patient suddenly became very ill with headache, stiff neck and temperature 103° F. Lumbar puncture revealed old blood in the fluid and 500 cells, predominantly polymorphonuclear leukocytes. The wound was only slightly reddened, but drained cerebrospinal fluid on the following day. Large doses of penicillin, streptomycin and terramycin were administered. The wound drained blood-tinged fluid at first, but only clear fluid after 10 days of drainage. Drainage was stopped at this time by taping it snugly over gauze. Cell counts on the cerebrospinal fluid varied between 100 and 2,000, with no steady tendency toward improvement. Streptococcus fecalis was cultured from the spinal fluid. At no time did the patient's neurological condition deteriorate. He was conscious and oriented on February 12, 1953, until he suddenly gave a single gasp and expired. The post mortem examination revealed gross thickening of the dura mater and leptomeninges beneath the explored area. There was microscopic evidence of healing leptomeningitis about the spinal cord. Intracranial involvement was minimal.

Comment on cases 1-4: In case 1 the diagnosis was complicated by severe residual paralysis following poliomyelitis. It is the only case in this series with a true Froin's Syndrome. Cases 2 and 3 are of special interest in that intervertebral disc rupture was suspected in both. However, certain points in the clinical picture were at variance and prompted myelography, which pointed strongly to the diagnosis of tumor. Case 4 teaches a number of lessons. Iodized oil may flow freely past a sizable tumor. Too, a catheter may meet little resistance when it is passed by a spinal cord tumor of some considerable size. Also, even when allowance is made for the difference in the segmental level and the vertebral level of a tumor, exploration may be too low. Infection is still to be feared despite the available antibiotics, the use of some of which might well add toxic factors of significance. Reoperation probably adds to the danger of infection which proved fatal in this case although it appeared favorable at the time of tumor removal.

Case 5. Meningioma: G. H. Y .,a white female aged 61 years at time of laminectomy, September 28, 1948, at Methodist Hospital, gave a history of unsteadiness in walking for 2 years with rapid progression for 1 year. Pain had not been present. The neurological examination revealed a marked spastic paraparesis with more marked ataxia and slowness than muscular weakness. A partial sensory level was present over the mid abdomen. Lumbar puncture revealed a manometric block and a total protein of 76 mgm. per cent. Two cc. of lipiodol injected at the same time showed a complete block at the superior margin of the 8th thoracic vertebra. At laminectomy a bony hard tumor, fixed to the posterior dura, but easily separated from it, was removed from beneath the 7th thoracic laminae. This was demonstrated microscopically to be a typical meningioma with a partial outer shell of true bone. The patient left the hospital ambulatory on the 12th postoperative day. When checked on April 10, 1953, she was neurologically negative and free from complaints.

Case 6. Meningioma: C. W., a white female aged 63 years at time of laminectomy, August 6, 1952, at Jefferson Davis Hospital, gave a history of progressive staggering and numbness for 4 years, total motor, sphincter and sensory paralysis below the lower abdomen for 2 years, and marked flexion position of the lower extremities with flexion contractures and decubitus ulcers progressive over the last year. Prior pain which circled the right lower abdomen had diminished. The neurological examination confirmed a complete lower abdominal motor and sensory level. Lumbar puncture revealed a complete manometric block and a "greatly elevated" total protein. One cc. of pantopaque, injected in the lumbar region, was shown to be arrested at the lower margin of the 9th thoracic vertebra. Operation was recommended and performed despite the poor prognosis for any neurological recovery. A typical meningioma, grossly and microscopically, was completely removed at the 9th thoracic vertebral level. Gel-foam was used to cover the defect where the involved dura mater was excised. Although no neurological recovery occurred, pain was completely relieved, flexor spasms were greatly diminished and all decubitus ulcers healed in a few months. Operation had been refused during the earlier period when the neurological involvement was probably at a reversible stage.

Comment on cases 5 and 6: In case 5 the hard calcified shell, adherent to the dura mater, is unusual in a meningioma. The fixation of the tumor was probably responsible for the absence of spontaneous pain or the production of pain by coughing or sneezing, despite its situation over the posterior roots. The tumor excision in case 6 was performed largely as a matter of principle since a total paraplegia had been present for 2 years. However, it resulted in diminution of the flexion reflexes and great increase in comfort.

Case 7. Lipoma: A. A., a white girl aged 19 years at time of laminectomy, April 3, 1942, at Me-

morial Hospital, gave a history of pain in the lower back, progressive numbness of the right leg and enlargement of a soft subcutaneous mass over the sacrum for 1 year. Neurological examination revealed a soft subcutaneous mass over the lumbosacral area through which could be palpated a defect in the lumbosacral fascia to the right of the midline. Marked paresthesias were present in all of the sacral segments on the right, but the paresthesias were unaccompanied by any motor or reflex changes. Lumbar puncture revealed normal manometrics and no block. The total protein was 23 mgm. per cent. X-ray showed spina bifida of the 5th lumbar laminae. Examination with lipiodol showed no obstruction. At operation a large portion of the fatty superficial tumor which merged with normal fat was removed. Through two separate fascial defects cords of fat and fibrous tissue extended through the dura mater into the subarachnoid space at the 5th lumbar vertebra and joined. The mass in the subarachnoid space did not involve the roots, but extended upward on the right to attach to the spinal cord at the 2nd lumbar vertebra. It was followed upward through a hemilaminectomy and divided, leaving a small part attached to the conus medullaris. The pathological examination of the specimens showed mature fat and fibrous tissue. Temporary bladder difficulty lengthened the hospital stay. When the patient reported, April 18, 1953, her only complaint was moderate numbness in the right posterior thigh. She has instructed in dancing, has married and has a 6 months old son.

Case 8. Lipoma: B. L. G., a white girl 18 years old at time of laminectomy, September 1, 1948, at Methodist Hospital, gave a history of progressive atrophy of the left leg with flexion of the toes and marked nocturia for several years. Neurological examination showed a heel gait on the left, 3.5 cm. diminution in the circumference of the left leg, absent ankle jerks and anesthesia of all the sacral segments. A large superficial lipoma, 12 cm. in diameter and 6 cm. thick, was present in the lumbosacral area. The x-ray examination revealed spina bifida occulta of all the sacral segments. Myelography demonstrated filling to the sacral tip and a filling defect on the *right* in the lumbar region. Spinal fluid total protein was 16 mgm. per cent. At operation the superficial lipoma communicated through the more superficial structures and the dura mater with the subarachnoid space. The posterior and anterior roots were so firmly incorporated in the intraspinal fat and fibrous tissue that it was possible to do little more than dissect the mass from its dural attachments. The pathological diagnosis was mature fat and fibrous tissue. A follow-up report, April 13, 1953, indicates neither progression nor improvement of the neurological deficit.

Case 9. Ependymal Cyst: N. M., a white girl aged 12 years at time of laminectomy, November 13, 1950, at Hermann Hospital, gave a history of weakness and spasticity, chiefly in the left lower extremity, since 1 year of age. Since 1948 numerous operative and non-operative procedures were carried out by the orthopedic service, before and after the laminectomy, because of severe flexion contractures of the hip and knee joints. The neurological examination revealed marked spasticity and weakness of both lower extremities with hyperactive knee and ankle jerks, extensor plantar responses, and marked contractures. No distinct sensory level was present and sphincter control was satisfactory. Lumbar puncture revealed no manometric block. The total protein was 16 mgm. per cent. Spina bifida occulta was demonstrated roentgenologically in the 1st, 2nd, and 3rd lumbar neural arches. Myelography, November 8, 1950, revealed a central filling defect at L-1 (Fig. 1) and a normal subarachnoid space otherwise from the sacrum to the cervical region. Laminectomy on November 13, 1950, revealed a smoothly lined cyst 1.5 cm. in diameter within the spinal cord at the 1st lumbar vertebra. It contained a clear congealed mass. The cyst wall reached the pial surface, and this portion was removed for biopsy. Convalescence was uneventful, but neurological testing was limited by the postoperative application of corrective casts. Despite every effort to correct them, the contractures have been progressive. Myelography, May 23, 1952, revealed a filling defect just below the operative site and a block of the pantopaque in the lower lumbar region (Fig. 1). This is difficult to interpret in view of the pathological diagnosis of benign ependymal cyst and the preoperative myelogram. Further studies are planned, and re-exploration will probably be indicated.

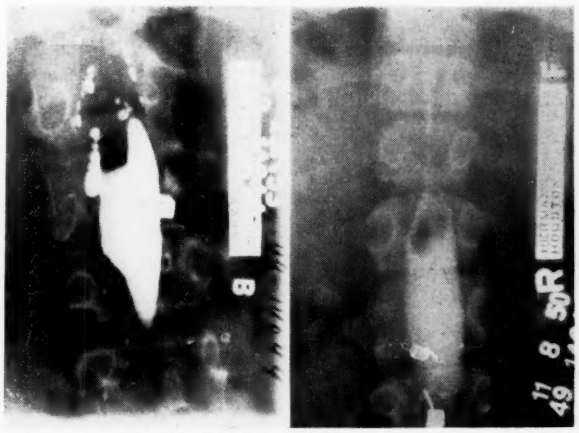

Fig. 1. Case 9. On the right is shown the constant filling defect observed at pantopaque myelography preoperatively, opposite the 1st lumbar vertebra. On the left are shown the filling defect at the lower margin of the laminectomy and the obstructive defect at the 3rd lumbar vertebra when myelography was repeated May 23, 1952.

Case 10. Dermoid Tumor: M. A. W., a white boy aged 9 years at time of laminectomy in September, 1952, at Jefferson Davis Hospital, gave a history of weakness in the control of his feet and incontinence of both sphincters, progressive since birth. Genital and saddle numbness had been observed. Neurological examination revealed saddle anesthe-

sia with involvement of the 5th lumbar and all the sacral segments on both sides, more severe on the right. Both ankle jerks were absent, and the right knee jerk was diminished. The bladder was readily palpable at the umbilicus. Lumbar puncture revealed evidence of a manometric block. Unfortunately, no cerebrospinal fluid total protein was recorded. X-ray examination revealed thinning of the pedicles of the 12th thoracic and 1st lumbar vertebrae and arrest of the upward flow of pantopaque at the lower margin of L-1. At operation an intradural dermoid tumor, verified by microscopic examination, was found. There was involvement of the conus and cauda equina, so that only a portion of the tumor capsule could be removed. When last checked, April 18, 1953, the patient showed no bladder distention and voided small amounts without hesitation and with a good stream. Evacuation of the large bowel was sluggish with incontinence only with the use of cathartics. The patient walked easily, but a bilateral heel gait was apparent. It is my opinion that improvement will continue over a period of months and that relatively complete rehabilitation is possible.

Comment on cases 7-10: In the two congenital lipomas more adequate surgery was possible in case 7 than in case 8, with resulting improvement over a period of 11 years. Case 9, the apparently benign ependymal cyst, has not benefited from surgery and now presents an unusual myelographic picture. Ultimate benefit appears unlikely. In case 10 a favorable outcome appears likely even though complete removal was not possible. The case illustrates how accurately a lesion at the level of the conus medullaris can imitate a lesion at the fifth lumbar vertebra. With compression at the conus recovery is likely to be less complete. Attention is called in these cases to spina bifida occulta in areas where it is exceptional, and to the importance of overlying abnormalities such as lipomas. Other cases than those presented have illustrated the importance of a congenital dermal sinus, a dimple or a tuft of hair.

Case 11. Osteochondroma: W. C. K., a white male aged 29 years at time of laminectomy at U. S. Naval Hospital, Oakland, December 11, 1945, gave a history of back ache for 18 months and spasticity with rapid loss of motor power in both lower extremities for 3 months. Sphincter control was greatly impaired. Neurological examination revealed hyperactive reflexes, positive Babinski signs and a partial sensory level just below the umbilicus. Lumbar puncture revealed no manometric block. The total protein was not recorded. X-ray examination of the spine was negative, but after the injection of 3 cc. of pantopaque there appeared to be partial arrest of the medium opposite the 8th thoracic vertebra. At operation a mass with a bony hard shell was found attached to the posterior surface of the body of the 8th thoracic vertebra in the midline. It was approximately 2.0 cm. long, 0.8 cm. wide and it projected posteriorly about 0.8 cm. It was completely removed extradurally by sacrificing the posterior articulation and pedicle on the left side. The mass had projected into the anterior part of the spinal cord, fixing the cord over it and making removal of the laminae far more traumatic than is usual. Microscopic examination revealed an osteochondroma. Motor paralysis was complete postoperatively, although there was partial sensory sparing. Later flexion reflexes became a grave problem. Motor function was improving 2½ months after operation when the patient was last under my care. Later alcohol was injected intrathecally, precluding further return of motor power. The patient has been rehabilitated as a paraplegic.

Case 12. Osteochondroma: I. M. G., a white female aged 61 years at time of laminectomy, August 31, 1949, at Jefferson Davis Hospital, gave a history of pain circling her abdomen for 8 years and spasticity, weakness and ataxia in both lower extremities, progressive for 1 year. Sphincter disturbances were marked. Neurological examination revealed marked spasticity of the lower extremities. Prior to operation a marked sensory level, 7 cm. above the umbilicus, was observed. Lumbar puncture revealed no manometric block, but the total protein was 80 mgm. per cent. However, only 2 months previously the total protein was 31 mgm. per cent. X-ray examination of the spine was negative; after the injection of 3 cc. of pantopaque partial arrest of the medium was observed at the 9th thoracic vertebra. At operation an osteochondroma similar to that in case 11 was found and removed in the same manner (Figs. 2 and 3). Postoperatively, motor power was lost except in the toes of the right foot. Motor power improved markedly in the right foot in the next 2 months and flickers of movement were possible in the left foot. Sphincter control had returned to a moderate degree. Flexion reflexes were a marked problem in this patient and did much to hinder rehabilitation. A report from the patient on April 15, 1953, indicates that she can stand by holding to the bed. Sphincter control is good during the day, poor at night. The patient is free from pain. She is dependent upon her wheelchair although she handles her body quite well.

Comment on cases 11 and 12: Grossly and microscopically the lesions in these patients were almost identical. From the surgical point of view these anterior fixed lesions offer the same difficult problem that occurs in calcified midline cervical disc herniations

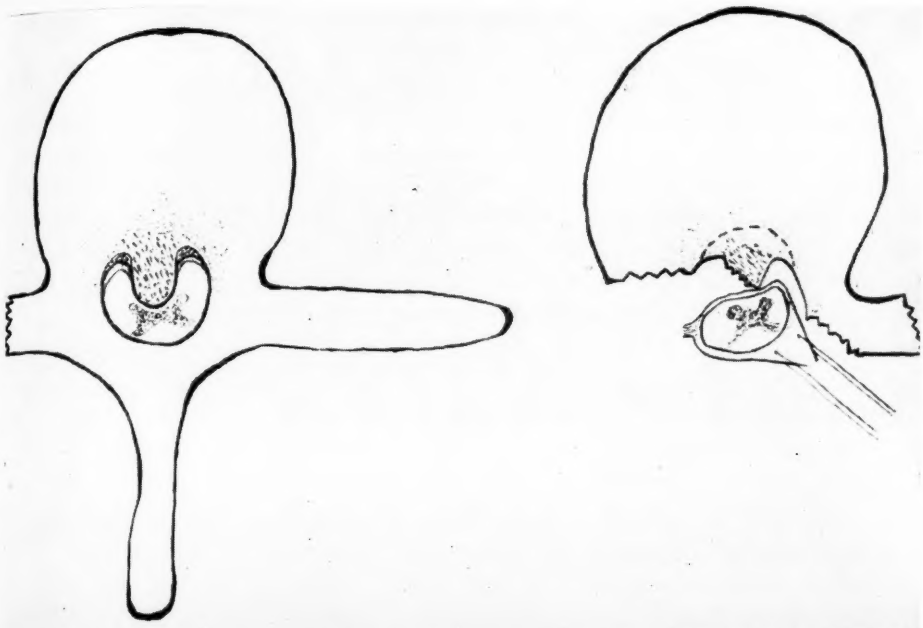

Fig. 2. Case 12. The spinal cord has been diagrammatically represented on the left as a crescent overlying the osteochondroma which extends posteriorly from the vertebral body. On the right the lateral surgical exposure by radical removal of the pedicle is represented. The broken line represents the area of excision of the tumor at its base.

Fig. 3. Case 12. Photomicrograph (hematoxylin and eosin stain, 270) of osteochondroma showing cartilage, bone and red bone marrow. The microscopic structure of the tumors in cases 11 and 12 was identical.

with spinal cord compression. In the narrow thoracic spinal canal the spinal cord becomes fixed over the anterior mass and forced directly against the laminae and ligamenta flava. In these 2 osteochondromas manometric block had not yet occurred and myelographic defects were only suggestive. Origin from the intervertebral disc is presumably excluded by both the anatomical position of the lesions and their microscopic characteristics. I have not observed reports of anterior spinal osteochondromas of this type in the literature. It is possible that they have mimicked degenerative lesions until late in their course, and that appropriate diagnostic tests, performed early, were not repeated.

Summary and Conclusions

Neurofibromas and meningiomas offer chiefly the problem of early recognition and localization. Even partial manometric block may occur late. The total protein of the cerebrospinal fluid is variable, even in neurofibromas which traditionally cause a marked increase. A manometric block, partial or

complete, is doubtless a factor in marked protein increase. It need not be present, as is demonstrated in case 2—total protein 371 mgm. per cent in the fluid above the lesion. Long duration of symptoms by no means excludes a diagnosis of spinal cord tumor. Even congenital tumors may become disabling only in middle or old age. The cerebrospinal fluid protein was normal in 3 of the 4 congenital tumors presented. Conservative removal has much to offer in the congenital group of tumors. The anterior osteochondromas offer the greatest technical problem of those tumors which can be completely removed. When a marked partial manometric block or a complete block is present, a small amount of iodized oil (½ to 1 cc.) is sufficient. Probably lipiodol is preferable in the demonstration of partial blocks because of its greater viscosity.

Psychogenic Factors in Migraine

(Report of a Case)

JOYCE S. LEWIS, M.D., and CLARENCE J. ROWE, M.D.
Minneapolis, Minnesota

It has been said that migraine headaches are the most common complaint of all civilized people and estimates place the number of American sufferers at 12 million.[9] This high frequency is reflected in the abundant literature on headaches in general and the migraine syndrome in particular.[1-18] The mechanism of headaches has been well formulated,[17] the hereditary, allergic and psychogenic factors have been emphasized. In recent years there seems to be increasing interest in psychogenic factors and personality traits in the migraine syndrome.[1,2,4,5,6,7,8,10,11,12,14,16,18] Most reports stress the importance of hostility and aggressiveness[5,11,12,16,18] and many portray such patients as obsessive-compulsive.[8,12,18] More recently two reports[4,13] have suggested that conflicts about dependency feelings seem important in some cases and one of these reports[4] further suggests that a variety of psychological conflicts may precipitate migraine.

Recently we treated a migrainous woman whose conflicts seemed to center about dependency satisfaction and repressed hostility. In addition, personality factors in her husband seemed to play a significant role. Since such an observation has not been previously mentioned in the literature, we thought it would be of value to report the case.

Department of Psychiatry, University of Minnesota, Minneapolis, Minnesota.

Mrs. J. P., a 35 year old attractive housewife, has had unilateral headaches associated with nausea, vomiting, and vertigo since age 3. For the previous two years the headaches had become so severe and of such long duration that they were completely disabling.

Family History: Her brother, her mother, an uncle and her paternal grandfather had similar headaches. Her father, age 62, a semi-skilled laborer, was described as a dominating man who ruled his family with an iron hand. The mother, age 56, has had "nervous troubles" most of her life and two episodes that were probably psychotic depressions (one after the birth of the patient and one 6 years prior to the patient's admission). The mother rebelled against the father's attempted domination and frequently argued with him about his sexual rights. As a youngster the patient many times tried to hide in her closet at night to escape her "parents' yelling." The patient favored her father, and some of her happiest childhood memories were of the trips they made together. She could gain sympathy from her father and control him with her headaches, but had to resort to temper tantrums to get her way with her mother. She was never close to her mother.

There are three siblings: a 22 year old sister who is in nurses' training and towards whom the patient acted as a mother, a 39 year old unmarried sister who still lives at home and takes care of the parents, and a 38 year old married brother who has been very close to the patient since childhood.

Personal History: The patient made a good adjustment to school and graduated with honors at age 17. She wished to continue her education, but lacked money and also had severe back pain which necessitated nearly two years of treatment with bed rest, casts, traction, and finally sacro-iliac fusion. After a year's work as a secretary she married her boy friend's roommate who was 10 years her senior. The boyfriend was a much more extro-

vertive dominate male who when he became serious in the relationship became too great a threat to her. After marriage they moved to a small community where he did very well in business. It was not long until her drive and ambition had gained for her "everything she had always wanted," including being at the top of their social group. They have two children, a girl age 13, who although apparently well adjusted has drive and ambition like her mother, and a boy 10, who is overly sensitive and has had episodes of vomiting and headaches. After the birth of her boy, she again had back pain and an intervertebral disc was removed. Four years before admission general market conditions caused their business to decline and an economy which was intolerable to her became necessary. Two years later her best friend's husband died suddenly and unexpectedly, following which she became depressed. This reaction seemed related to the patient's guilt and rage at this man's sexual advances towards her during the previous month. A few months later her brother, who had worked in their business, had to leave because of the business decline, taking with him his 2 year old son to whom the patient had formed an unusually close, neurotic attachment. It was only after these events that the attacks became severe and incapacitating. All conceivable treatments were tried, including courses of intravenous histamine, cortisone, DHE, and other ergotamine preparations. Finally the middle meningeal artery was tied and alcohol spread over the dura. Because none of the treatments seemed to help and because she was developing a dependence on dolophine and barbiturates, lobotomy was being considered.

The patient and her husband were totally resistive to psychiatric referral and only accepted it when other specialties refused her further treatment.

Patient's Personality: She has always been a very active, aggressive, rigid, extrovertive, narcissistic, hard driving person who "can get three days' housework done in one day." She has to have everything "just so," especially in her home and is very bothered by dirt anywhere. She makes fast and sure decisions and is very impatient with others who do not, e.g., her husband. She goes to any length to complete to perfection any project she starts. She likes to be in control of her environment. Her husband describes her as somewhat aggressive and "oversexed."

Husband's Personality: The husband is an outwardly rigid, hard driving person without much warmth, who poorly hides a great deal of hostility. Most of his married life he has had a duodenal ulcer, but was surprised that during his wife's illness he has no ulcer symptoms. He had to assume responsibility at an early age and feels that emotional illness is "just lack of will power." He recognizes that his wife has dominated the home up until her illness and states he is not going to let her do that again. During the course of her treatment he has attempted to play the role of therapist and has in various ways encouraged her illness, e.g., sabotaging any attempts of doctors to set limits with her about drugs. He was especially threatened by his wife's free expression of emotion and could not tolerate any hostility from her. Therapy was attempted with him, but he was so disturbed by the free expression of his feelings that he could not participate.

Course of Treatment: Our goal in therapy was to return the patient to her level of minor symptoms two years earlier. Therefore, throughout treatment deeper interpretations and probings were avoided. The treatment can be divided into three phases corresponding to the predominating symptomatology:

Phase *one* corresponds to the first hospital admission when the patient resisted involvement in a psychotherapeutic relationship and denied any personal difficulty. Treatment was directed to her narcotic dependence, her electrolyte balance, and her anuria (from vomiting and anorexia). This involved a severe struggle with the patient as she used every possible means to control the situation and gain her wishes. After a period of a week without a headache she was discharged and offered outpatient interviews. Since this hospitalization headaches have never been her main complaint.

During phase *two* the patient dropped many of her intellectual defenses, experienced more feeling in life situations, and became aware of her unconscious feelings of dependency and hostility. This produced anxiety to which she reacted with two extremes of behavior: (1) depression and suicidal gestures, and (2) mildly destructive, "acting out" behavior. During this phase she was hospitalized twice.

Phase *three* began when the treatment was changed from a permissive-interpretive approach to a limit-setting technique in which she was expected to assume responsibility for her behavior. For the last eleven months during which time she has been given continued outpatient support, she has been adjusting with only minor symptoms.

The total interview time was approximately 80 hours.

Discussion: The basic problem in this patient is unresolved dependency. This seems related to the period of neglect in infancy during her mother's psychosis, a somewhat cold, narcissistic mother, intense sibling rivalry, and an unhappy, unstable home situation. Early in life, she began to deny strong dependent needs, and although she felt intense rage at minor slights, she could not give vent to her feelings either because of their overwhelming nature or for fear of losing the little approval she did receive. Repression and reaction formation were the family's approved methods of dealing with feelings. Complete female identification did

not occur and the patient has shown great resentment at having to assume the feminine role. This resentment she has handled by reaction formation: being a "perfect" wife and mother and claiming "perfect" sexual adjustment. In the psychoanalytical sense, she has not satisfactorily resolved her Oedipal situation. In her personality adjustment we found both compulsive and hysteroid features.

One might make the following dynamic formulation:

$$\text{Dependency} \begin{matrix}(\text{threatened}) \\ <\text{aroused}> \\ (\text{frustrated})\end{matrix} \longrightarrow \text{Hostility (unexpressed)} \longrightarrow \text{Migraine}$$

In this "perfect" marriage the character difficulties of both marriage partners balanced each other. They both dealt with hostility by repression and reaction formation. Neither could tolerate conscious dependency gratification: the husband suffered from duodenal ulcer; the wife from migraine. During treatment the husband became a very significant negative factor because of his complete inability to accept increased free hostile expression from his wife, and his apparent need to keep her ill. (We have seen two other cases of migraine syndromes in women in which the husband played this same antitherapeutic role.)

Therapy in this patient seemed first of all a situation in which she could have a safe dependency relationship while developing a better understanding of her unconscious feelings of resentment. During therapy she was able to practice release of these resentments in a protected situation and with this release noticed abortion of her headaches.

Summary: We have reported a case of migraine syndrome in a 35 year old married female. Unexpressed hostility and unsatisfied dependency feelings, as previously reported by others, seemed paramount. A third factor of importance in this case was the personality of the husband.

It is suggested that psychopathology in the spouse may be of etiological importance in migraine and deserves further study.

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